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Contagious Lymphangitis.

Although this disease has been recognised for some years, and was well described by the late M. Nocard, it has only been seen in this country very recently.

Mr. J. A. W. Dollar gave the profession an early translation of Nocard's work, so we may presume that if the disease had been met with here it would have been diagnosed. It is quite possible that horses in London may have been seen suffering from contagious lymphangitis and destroyed as glandered.

Since the war in South Africa the return of horses to this country has introduced the disease, and at the present time cases exist both in London and Dublin. To any ordinary observer the cases would suggest farcy. The enlarged lymphatics, the more or less swollen leg, the small ulcerating sores, are all symptoms such as we have often recognised in ordinary cases of farcy. Possibly if we saw more of the disease we might differentiate between it and glanders-farcy, but without a knowledge of the papers contributed by Mr. Dollar and by Mr. Parker the practitioner might easily be mistaken.

There is a difference between farcy buds and the sores of contagious lymphangitis, but it is most evident to those who have experience of both conditions. If we had one leg affected with the new disease and the horse rubbed the parts with the opposite leg, a close resemblance to the clinical appearances of farcy would be produced.

Fortunately there is a method of diagnosis—in fact a negative and a positive method. The contagious lymphangitis does not react to mallein, and the pus when examined under a microscope discloses crypto-cocci of such extraordinary size that it is impossible to confound them with any other pathogenic cocci.

This disease is, we are told, curable, but cure requires time and entails expense. It is contagious, and possesses a very long period of incubation, so that it may break out months after the day of infection.

The late M. Nocard warned his Government against the danger of importing many foreign diseases which might cause great national loss. The warning was of interest to France, but how much more so to Britain which directly communicates with all the world. Some diseases cannot be introduced here owing to their short period of incubation, and some are of little importance because they do not spread in our latitudes.

This contagious lymphangitis is already introduced—it may spread. What are the Board of Agriculture doing to protect o r home stock?

IMMUNITY AND DISEASE AS FACTORS IN THE EVOLUTION OF ANIMALS.

By Gerald R. Leighton, M.D., F.R.S.E.

Professor of Pathology, Royal Veterinary College, Edinburgh.

(A paper read before the Scottish Metropolitan Veterinary Association, November, 1903).

I wish to deal in this paper, very briefly, with an aspect of the life history of the domestic animals especially, which seems to me to have a very direct bearing on the problems of modern veterinary science. It is an aspect which seems to have had singularly little attention paid to it by veterinarians in the past, and which even at the present time does not claim a large share of the attention of the profession. But in the sister science of human medicine the question to which I wish to direct your attention is beginning to occupy a very prominent position, and to excite some very animated controversies. It may, therefore, be worth while to look at the matter from the veterinary side and see if it does not suggest to us some food for thought.

Put in the shortest possible terms, the question is:—To what extent and in what directions has disease acted as a factor in the evolution of animal species as we now see them?

Everyone nowadays who knows anything of the facts agrees that our animals have become what they are by means of a slow and gradual process of evolution acting over a long period of time. And if it be asked what has been the broad principle which has guided this process of evolution, and which has resulted in the modern animal species, the answer given in general terms would be that of the well-known formula: A continuous struggle for existence in which a natural selection has allowed of the survival of the fittest only. That answer is undoubtedly the truth, even if it be not the whole truth. As a matter of fact it implies the whole truth, leaving the details unstated. The details are the special factors concerned in producing the "fittest" animals, and while some of these factors have received great attention and due weight, others have been comparatively neglected, and—as I think—some of the most important.

That this is really the case will be realised if we consider for a moment the answer that would be expected and given to a question, such as for example, What has been the process of evolution in the case of the horse? A candidate for examination or any other individual replying to such a query, would in all probability regard the animal exclusively from

the morphological standpoint. He would describe the horse as having sprung from a primitive five-toed ancester, such as Phenacodus. As time went on, in the Eocene period—a period of disturbance from a geological point of view, and as the marshy land gave place to a harder surface, there appeared now and then individuals in which the first toe lost its phalanges. These variations, at first occasional, became constant in the descendants, until the character was a fixed one. The loss of this toe being an advantage in speedy movement gave the individuals possessing it a greater chance of survival in the struggle for existence. Similarly in the middle Eocene, animals like Orohippus were produced in which the splint bone of the first digit was smaller or absent, this in its turn becoming a constant character. Still later the three-toed tribe arose from the four-toed by a further variation which was transmitted to the offspring conferring an advantage, and by a similar process in due course the one-toed animal became established. Along with these modifications in digits were corresponding changes in muscles, teeth, etc., culminating in the modern horse as we see it.

Such are the lines upon which are commonly traced the evolution of the horse. I have, of course, omitted detail, my only object here being to point out that structural modifications are the factors upon which all the stress is laid, and I quote the case of the horse because in that animal these successive changes are best known.

I say then, that in the minds of most of us, the process of evolution is associated almost entirely with variations in the *structure* of animals, which enabled them to adapt themselves to the altering conditions of their environment, and which resulted in the survival of those individuals which varied in a manner advantageous to the particular environment of the age in which they lived.

But the morphological aspect, though of fundamental importance, is not the only one from which we may regard an animal, nor are morphological variations the only factors concerned in the evolution of the species. Structure alone does not complete the account of the individual nor explain entirely how he came to be what he is. There have been other factors contributing their share to the elimination of the unfit and the survival of the fittest, and of these the factor of disease must, in my opinion, have held a leading place.

One of the most striking features in the specific characteristics of the domestic animals is the great divergences they exhibit in the matter of susceptibility to disease. Some are naturally immune to this condition, others to that; all exhibit some reactions to disease peculiar to their own species. Some of the most familar instances of this are the following:—Horses are very susceptible to glanders, it is the most fatal equine disease; horned cattle are immune to glanders; goats and sheep are partially immune. But some horses are much more resistant than others.

Cattle and sheep are very susceptible to anthrax; but Algerian sheep are almost immune to anthrax, and horses are not nearly so susceptible.

Tetanus, although it can be inoculated into other animals, affects chiefly man and the horse.

Tuberculosis, which is the most widespread human disease, exhibits great variation in animals; cattle are most susceptible, it is far less common in sheep and goats, while many other animals are intermediate in their liability.

Field mice, which are susceptible to glanders and tubercle, are immune to strangles. House mice and white mice resist tubercle.

Finally, man is susceptible to a number of conditions which do not affect the lower animals, while he is immune to some diseases which affect them. Innumerable instances such as those quoted might be added, but these will be quite sufficient to show that along with structural evolution in species, there has been an evolution as regards disease, which is no less striking.

It is by no means so easy to read the history of the past ages of disease in animals, as it is to decipher their ancient structure. The records are not obtainable; we have no fossil diseases. Veterinary literature does not go back far enough to help us. But there is one method, and as far as I know only one, by which we may endeavour to estimate the part played by disease in the past, and that is by observing its mode of action at present. As far as we know the general laws of biology are the same now as they were in ancient times, though they may be acting in somewhat different circumstances. If therefore, it is desired to estimate the extent to which disease has affected evolution in the past, and the manner in which it has acted, it is necessary to observe its action in the more recent periods of which we have some definite knowledge. In other words, if we can discover the part played by disease in the history of the human race, we shall get some idea of what has happened in the case of the lower animals.

It so happens that there is in existence a mass of facts bearing on the problem from the point of view of human races, and while I have no desire to dogmatise concerning the interpretation of these facts, I do wish to mention some of them in the hope that they may add an increased interest to our study of comparative pathology. We may regard diseases in this connection as of two kinds:

(1). Those against which the patient can acquire some degree of immunity (*e.g.*, smallpox, strangles), cattle-plague, contagious pleuro-pneumonia, and black-quarter.

(2). Those against which no resisting power can be acquired (*e.g.*, consumption), tuberculosis, erysipelas.

In the first class, one attack leaves the patient partially or completely immune from a further infection, while in the second class a first attack weakens rather than strengthens against subsequent infection.

Now apply the doctrine of the natural selection of those individuals which are best fitted to survive and observe the result. Note that "though one attack of certain diseases usually confers immunity on the individual, yet, in such cases, the race never attains to immunity. Each succeeding generation

remains as susceptible as the preceding."* A horse recovered from strangles may remain immune for life, but its offspring are as susceptible as the parent was before the attack. "Thus Englishmen are as susceptible to infection to measles as Polynesians,"* although individual Englishmen have for generations acquired immunity from measles from an attack in childhood. "But since measles weeds out those who cannot recover from it (*i.e.*, those who cannot *acquire* immunity against it), the direction the evolution takes is towards an increase of the *power of acquiring immunity*."* That is to say those individuals which had inborn in them the greatest resisting power are the fittest to survive, and do survive, and transmit their resistance to their descendants, the result being that the race increases its power of acquiring immunity against measles, which in time thus becomes a comparatively trivial disease for that race. But if introduced into a new race, as the Polynesians, amongst whom there has been no selection of the fittest, the disease may wipe out a whole tribe. "For that reason, though Englishmen are as susceptible to infection by measles as Polynesians. they recover from it much more easily."

We thus see that natural immunity cannot arise in animals by transmission of acquired immunity of the parents, all that happens in that case is that the race increases its power of *acquiring* immunity.

(2) Under what conditions, then, is natural or innate immunity evolved, or does it tend to be evolved? We see that it exists in many animals against various diseases. How could it have arisen? Look again at the history of the human race and see what has happened. "For very many centuries consumption has ravaged the Old World, especially such crowded parts of it as England. But Englishmen now increase and multiply in cities and towns, the natural breeding places of consumption; whereas, under like conditions the inhabitants of the New World, where consumption was unknown till recently, perish. When infected with consumption by white men they tend to extinction everywhere, even in rural districts. Plainly there has been great evolution, but as plainly it has resulted solely from the survival of the fittest, and not in the least from the transmission of acquirements." The conditions under which a large proportion of our population live, mean that very many are immune to tubercle, naturally immune. This innate immunity has been evolved in the race as the result of long exposure to the disease. It is never acquired by the individual who has an attack. And therein is the answer to the question we asked. The answer is that "The only diseases against which inborn immunity is, or tends to be, evolved, are those against which the individual cannot acquire immunity."

In the first class of diseases noted, "When immunity against disease can be acquired by the individual, then the *power of acquiring it* is evolved in the race by Natural Selection. When it cannot be acquired by the individual, when one attack weakens rather than strengthens, then inborn immunity is evolved in the race. In the one case the capacity to *recover from* infection is evolved; in the other the capacity *to resist* infection. In both cases the evolution proceeds wholly on lines of Natural Selection."† So the Englishman has increased his power of recovering from measles and his capacity to resist the infection of tuberculosis.

These same arguments apply equally to other diseases which have from time to time decimated portions of the human race, *e.g.*, small-pox and malaria. "For thousands of years diseases due to micro-organisms have been the main cause of human elimination, and therefore of human evolution," and it is impossible to avoid the conclusion that the same must be true of the world of lower animals.

The result of a careful examination into the history of those races which we know to have been subjected for centuries to the action of infectious diseases, enables us to formulate a general statement or law, to the effect that every race is capable of resisting infectious disease just in proportion to its past experience of that condition. Applied to animals, these considerations would suggest the following ideas.

In a case such as strangles, in which the horse having suffered once may be subsequently immune, the evolution in that animal species is in the direction of increased power of *acquiring* immunity; so that fewer and fewer cases will have serious results, but successive generations will continue to be attacked. In a disease like tuberculosis, in which the animal once attacked shews no tendency to subsequent immunity, the weakest individuals are eliminated in course of time by the disease, and the evolution is therefore in the direction of increasing the natural resistance to infection, because in each succeeding generation those survive which can best resist. That is to say the tendency of evolution here is in the direction of producing an inborn immunity. So that in course of time the Anglo-Saxon race, and those domestic animals which have been most exposed to the ravages of tuberculosis, may be expected to become naturally immune to tubercle. As a matter of fact, this has already taken place to a certain extent in our own country, where tuberculosis does not now play the same havoc with our population as it does when introduced into a people for the first time.

To sum up in a word the argument here advanced. The problem is to account for the different degrees of susceptibility and immunity exhibited by man and animals to diseases. (With the question of the origin of disease or its species distribution we are not now concerned.) We find certain diseases affect a certain number of animal species, some species more, some less, some individuals more than others. We find that in those diseases in which the patient can acquire some individual immunity as the result of having been attacked by the disease, the result of natural selection is to evolve a race which is no longer decimated by these conditions, in other words, the race *increases its power of acquiring immunity*. Secondly we find that in those

* *Alcoholism*, Archdall Reid, p. 36.

† *Ibid*, pp. 35, 36.

diseases in which the patient does not acquire an immunity after attack, the result of natural selection is again to produce a race no longer decimated by the disease, but in this case because an inborn immunity has been evolved. The only diseases against which this inborn immunity is, or tends to be evolved, are those against which the individual cannot acquire immunity, *e.g.*, Tuberculosis.

It is, of course, impossible in one paper to even mention any of the very many factors which may assist or interfere with the working out of such processes in any given animal species. My only purpose here is to draw attention to the great general principles involved, and the evidence which seems to me to bear upon the point at issue. The further discussion of how other factors would effect these principles, factors such as in-breeding, artificial selection, civilisation and all its consequences upon animal life, etc., etc., would form a most fascinating topic, but one which I must not enter upon to-day.

Now, gentlemen, I have led you to the threshold of what—to me at least – is a most fascinating subject, but only to the threshold. There I must for the present leave you, in the hope that I have at least suggested to you some thoughts which will lend an additional interest to the many obscure problems which veterinary pathology still offers for solution.

FRACTURE OF THE OS CALCIS.

By E. Wallis Hoare, F.R.C.V.S., Cork.

A well-bred five-year-old huntress, while being "lunged" over banks, slipped when jumping off a bank, and on rising was found very lame on the off hind leg. She was brought with great difficulty to her stable, and my attendance was requested next morning.

The symptoms presented were great pain and uneasiness, the animal moving round the stall with the hock strongly flexed, the tendo-achilles loose, the *tuber calcis* displaced forwards, crepitus was easily detected, and a sharp portion of the posterior edge of the tuber calcis had penetrated the skin slightly at the back of the hock. The animal had got down in spite of the attendant shortly before my arrival, but quickly got on her feet again.

I advised that she be destroyed, which course was adopted.

On examining the part I found evidence of much bruising of the tissues. The tuber calcis, carrying with it portion of the an terior surface of the body of the bone, was separated and carried forward. The fracture extended from the upper part of the posterior surface of the tuber calcis, downwards; and on the external aspect there was a triangular shaped fracture which separated a portion of the body containing the articular facet for the cuboid bone. A small portion of the articular cartilage was detached from one of the articular facets for the astragalus.

It will thus be seen that thebone was divided into three pieces. The accompanying photo shows the direction of the fracture.

SURGICAL TREATMENT OF STURDY IN CATTLE.

Within the last twelve months I have had occasion to operate for cyst in the brain of six cattle, of various ages and sex. The instruments used were as follows: Mannington trephine ¾ inch; possibly a smaller size would have been better, but that was the only one I had, and the remuneration to be had for the operation did not justify an expensive outfit, 3-16ths in. trocar and canula, crochet pin, ordinary operating instruments, absorbent wool and disinfectant. I also carry a small quantity of pitch with me in case there may not be any in the farms. The instruments I put in chinosol or carbolic acid solution whilst I get the patient ready for operation.

I may say that I have operated in different ways on sheep and cattle in years gone by, but not with the same success as in the cases that I am about to record.

I did not take notes unfortunately of the cases as I was doing them, but I can call to mind all the important facts.

Case I.—Oct. 4th, 1902. Bullock about twelve months, unable to stand, in very low condition, turning to the right before he got too bad to stand, no softening of the bones of the head, but on tapping the forehead a distinct solid sound was perceptible on the right side compared to the left, and slight bulging of the bone.

Secured, I cut the hair off as close as possible with a pair of scissors, and thoroughly washed the forehead with soap and water and disinfectant (creolin). I made crucial incisions a little to the right of the median line, midway between the orbit and top of the pole, dissected back each of the flaps and scraped the tissues off the bone. I penetrated the bone with a gimlet and trephined the bone, which was quite hard, and the piece cut was elevated intact with the centre piece of the trephine. When the brain was exposed a portion of the cyst could be seen protruding through the hole made by the gimlet in the coverings of the brain. I seized the cyst with the forceps and letting out the fluid the whole was removed without any difficulty.

I had the wound washed with chinosol solution, removed blood, dried with wool, and closed by inserting two cross silk sutures through the angles of the flaps, finishing by placing a small pitch plaster to serve as an antiseptic and protection to the wound.

I left instructions to have the plaster removed and the part washed should any signs of suppuration present themselves, and the stitches cut off in five or six days. I did not see the case again for some months until it was exposed in a fair, but was told by the owner that it was soon on its legs and feeding.

Case II.—Jan. 19th, 1903. Bullock, older than the first, in good condition, turned to the left, very wild, would run against the walls and roll over when approached, no softening of the bone, but a difference in the sound was noticeable to the bystanders when tapped.

I proceeded in the same manner as with the first case, but unfortunately the trephine went through the bone with a jerk, injuring the brain and bursting the cyst; evidently causing a great deal of pain for the bullock gave a loud bellow. Inflammation of the brain set in, but not of a septic nature as far I could make out from a description of the wound given by the owner, the bullock becoming very violent, throwing himself about, broke one of its horns in struggling. The owner considered the case hopeless, slaughtered it some days after the operation.

Case III.—Feb. 12th, 1903. Bullock, similar symptoms to the second, no softening of bone, no bulging; with perceptible difference in the sound of affected side.

In this case the cyst was deep seated. After trephining I cautiously inserted the trocar about half an inch, withdrew stilette, but no fluid exuded through the canula. I had to push the trocar from 1¼ inch to 1½ inch before finding the cyst. I pumped the fluid out and tried to hook the cyst with the crochet pin but failed. He soon recovered, and was sold off before he showed any symptoms of the cyst refilling.

I saw this case about nine days afterwards. The plaster had come off, stitches intact, and the wound had evidently healed by first intention.

Case IV.—June 4th, 1903. A heifer, at the same farm as Case III, similar in many respects to the last, cyst on the surface, but the bone appeared to be more brittle than the other cases. The trephined piece was broken into several fragments. Regained the power to control his movements in a shorter time than Case III. Complete recovery. The respective age of the last was from twelve to fifteen months.

Case V.—March 17th, 1903. Heifer, younger than the rest, turning round, slight bulging of the bone, but no softening. The fluid exuded through the gimlet wound, and the cyst removed after trephining. Rapidly recovered.

Case VI.—Nov. 16th, 1903. Bullock, about two years and three months old, in good condition and feeding occasionally, would walk round the box to the left, grazing the sides with the right. There happened to be a recess in the wall into which he would keep pushing his head and remain in that position for hours if not moved. In his struggles the right horn was broken off close to the head.

I gathered from the symptoms that the cyst was on the left side and near the centre of the front part of the brain, no softening of the bone, but a slight bulging was perceptible to the feeling and sight when the hair was removed. Tapping in this case did not assist me in locating the cyst, in fact the sound was if anything more solid on the right. The bulge was a little to the left of the median line over which I had prepared to trephine. When the gimlet was withdrawn the fluid squirted out some distance. The idea occurred to me of trying to get the cyst out without trephining. I enlarged the hole made by the gimlet by twisting the angular end of the trocar a few times. I managed to hook the cyst with the crochet pin and gradually ex-

tracted the whole of it. I had made the incisions longer in this case than usual, so I put a silk suture in each incision and two in the corner of the flaps – six in all. He moved the next day almost normally, and looked about intelligently.

November 24th.—Completely recovered; turned amongst the rest of the cattle.

R. JONES.

Towyn.

PARTURIENT APOPLEXY.

I have read with much interest the reports of veterinary surgeons in the country who have treated this affection *à la Schmidt*, and thought that a few words of mine would be of some interest to your readers.

In the first place, dairy cows near London are largely fed upon brewers' grains, and in dealing with animals whose constitutions have been so abused by this treatment, we must remember that sooner or later the machinery must give way. Cows were never intended to be converted into milking machines and consumers of brewers' grains, any more than a man's system was intended to be continually saturated with alcohol. Years back it was customary to buy cows at the fourth or fifth calf, grain them and force them for milk as much as possible, and then sell them out fat. A dairyman would scarcely think of calving a cow under this feeding; in fact, I hold that two years of this treatment is sufficient for any cow.

What do we find to-day? Cows are calved with the first calf, grained and forced for milk year after year until the system collapses, which in a great many instances it does after calving, or they fall victims to tuberculosis. Treatment *à la Schmidt* is in my hands of no use to animals of this description suffering from parturient apoplexy. Whisky and gruel, practically *ad libitum*, are the only things which will keep up the heart's action, in fact taking all in all, I have found the odds are decidedly in favour of the pole-axe, at any rate, it is quite certain that we can never replace worn out duplicate parts. Some day perhaps we may discover the microbe of wear and tear, but until this germ is found and isolated, we must render to the omnipotent the the things which are the omnipotent's.

As to the treatment of Devon cows naturally fed suffering from this malady, it is well to be careful, particularly if the owner is a true Devonshire man with no admixture of Anglo-Saxon blood. A client of this description is in my hands very difficult to satisfy either with attendance, advice or charges, and a few hints on handling Devonshire men would be thoroughly appreciated by myself. If a client of this description was inclined to use the pole-axe I should be very dubious as to advising otherwise, feeling perfectly certain that if the animal died under Schmidt's treatment there would be some grumbling when the account went in, although it is always best to see that the animal is destroyed before the practitioner takes his leave.

Lastly, as to my experience near London. I am bound to admit that the knife is the most satisfactory and profitable remedy in true parturient apoplexy, unless a practitioner has private means and ample leisure and can, so to speak, afford to be a philanthropist; although ready to admit that in treating Devon cows a practitioner has a constitution second to none to deal with. Graining and forcing shorthorn cows is ruining not only the breed but their progeny.

GEORGE UPTON.

ABSTRACTS FROM FOREIGN JOURNALS.

THE PROPAGATION, INCREASE, AND DEVELOPMENT OF TRYPANOSOMES IN THE BLOOD OF ANIMALS ILL OF SURRA.—By A. BRAUER, Government Veterinary Surgeon.

(Continued from page 332.)

These parasites now having attained the dimensions of $\frac{1}{3}$ to $\frac{1}{2}$ the diameter of an erythrozyte, commence amæboid movement as may be very beautifully seen by the lesions caused in the red blood corpuscles. At first in coloured preparations sharply contoured and sharply limited unstained defects may be recognised in the blood corpuscle. Also considerable deviations from the normal form may be noticed in this body as a result of parasitism. Most frequently one sees the edge of the blood disc bent out of the even, also forms designated by pathologists as "hat brims" may be seen; further I have frequently noticed a cuplike shape of the blood corpuscles in the cavity of which the blue coloured structure may be seen. In coloured preparations the above related defects explain the form of the growing spores. Frequently one sees if two such spores have at the same time selected one blood corpuscle as their host, that this now resembles a ring through which a plain or wrinkled ribbon has been drawn.

These defects are not, as at first one might surmise, favourably influenced by the processes of fixing and staining, one can see them very beautifully in fresh blood, and I have met with them in greater or less quantity even after the stage of the illness.

It is extremely disappointing that in the whole of surra literature, with one single exception, these gross lesions of the red blood corpuscles have not been mentioned. Rankin alone describes them in *The Veterinary Journal*, London, 1891, p. 400-401. He is, however, on quite a false scent, for he believes that a bacillus is the cause of surra. He has also seen the cocci-like forms in the red blood corpuscles, and he describes very clearly the changes taking place in it.

After the future trypanosomes have left the blood corpuscles one meets them in large quantities free in the blood as bodies of irregular, indeterminate forms of about 2-4μ diameter. One can now discern quite plainly the size and colourability of the two different kinds of free parasites. The smaller of about 2-3μ diameter colour themselves violet with older methylene blue solution, and at times an excentric little white point may be recognised. The larger of about

3 to 5μ diameter colour themselves pale blue, are granulated, and have a somewhat larger clearer spot. Both commonly show at times poikilozyte form. The offshoots occurring thus, produced by amœboid movement, at times lie diametrically opposite one another, and then forms arise which a superficial consideration and some imagination might mistake for youthful trypanosomes. One of the large and one of the small bodies now approach one another, and then the larger begins to take up the smaller into itself like a phagocyte. This pseudo-phagocyte grows a little by this. The previous clear spot in him is added to the circumference, and the little violet bodies increase somewhat in size through intussusception. The whole now has about the superficial area of a red blood corpuscle.

Now the body begins to lengthen, at one end it is truncated and there is a vacuole; at the other end the pale protoplasma is drawn out to a gradually lengthening fine flagellum. The little violet body lies about where the future nucleus arises, and it is differentiated plainly from the remainder of the granulating body of the parasite by its violet, diffuse colour. A centrosoma is at times recognisable as a very small, pale black point in the neighbourhood of the vacuole.

What becomes of the disunited and to a certain extent unencumbered forms I have not ascertained with certainty, and I will not therefore give my opinion concerning them. The more the young trypanosome now lengthens itself the more slender the body becomes and the smaller the vacuole at the blunt end. Finally the vacuole vanishes completely as well as the violet provisory nucleus.

In this form I have most frequently encountered the parasites in the blood. A so-called "undulating membrane" does not detach itself from the remaining parasitic body; there is, however, a peculiar stratification of dark coloured granula created in the body as if a special membrane was present. The whole flat formed parasite has a somewhat thick membrane on its long side so that a frontal section would give a wedge-shaped surface, and the surface of the break a surface similar to the blade of a knife.

The anterior sharp end of the trypanosome is stained somewhat darker than the blunt hind end, whilst the latter in certain stages takes up no colouring stuff, but can only be recognised as a deep blue coloured contour. The enclosed clear space in this contour is at times crossed by a coarse network of the finest blue lines.

The thus described course of development of surra trypanosomes reveals the fact that one may look in vain for little trypanosomes in the blood as many authors have seen and put on paper. The typical parasite is in consequence of its peculiar development only liable to small fluctuations of size and form, and the little narrow structures that occur and which have been ascribed to long stalking are not trypanosomes but only phantasy forms which have fulfilled their object up to now, namely, to fill up the void that occurs in the increase of trypanosomes. —*Ex. Berliner Thier. Woch.*

G. M.

ON CERTAIN SEPTICÆMIAS AND SOME OTHER INFECTIONS OF YOUNG ANIMALS*

Discussion. (Abstract)

Mr. James: I have seen a large number of cases, and my experience coincides with that expressed by other gentlemen. We always thought it was a contagious disease, but farmers and owners of stock thought at one time it was due to the cow's milk, because the disease breaks out always within a week, and generally within two or three days. I do not believe that the scour which exists in a three weeks' old calf is the same as that in a two or three days' old calf. We tried years ago to try and better the conditions that surround the cows. That failed. The owners very soon perceived that if they removed the calves to different surroundings they might succeed. They took the calves from the cows immediately they were calved, put them into clean houses, and fed them on the milk of cows which had calved for a few weeks. Those calves lived. They attributed that at the time to the milk. I soon perceived the source of the infection, which I believed at that time was on the outside of the teat; the cows teat had become foul from the infected excreta of the diseased calves, and as soon as the calf had once sucked the cow it became infected. If the calf was removed without sucking the cow it lived; pointing out that infection was somewhere. I have had an experience similar to that of Mr. McGavin. When we could not do anything else the cows were turned into the fields, and the calves all lived. It mattered not what other treatment was adopted, it failed, but if the cows were turned into the fields all the calves that were brought forth after that lived. That pointed strongly to infection somewhere. I am not competent to express an opinion as to the portal of infection, whether it was the navel or the mouth; I have told you just what occurred in my own practice, and you must draw from that your own inference.

Mr. D. Pugh: In 1894 I had charge of a stud of mares and, being interested in temperatures, I got the manager to take the temperature of every foal immediately it was born. The first six were all right, but the temperature of the seventh foal was 105 immediately after it was born. The manager, thinking there was something wrong with his thermometer, took the temperature again, and it came out at 105. I instructed him to take the temperature of the mare as well as that of the foal, not because I anticipated any disease, but simply because I was interested in temperatures.

A Member: What were the temperatures of the other foals?

Mr. Pugh: 101·5, which I have always found to be the normal temperature of a foal immediately it is born. The manager wired for me, and on my arrival he told me that the foal's temperature was 105. I said, "What is the temperature of the dam?" and he said, "101·5." I asked, "Is your thermometer all right?" and he said, "Yes." I then said, "You have not used an enema for the foal, I suppose?" and he replied "No." I took my own thermometer, and it registered 105 and a little over. The foal went on very well for a little while, and then a violent scour took place and it died within 24 hours. We had another foal the next day and the same thing happened. The temperature at birth was a little over 105, and the mare's temperature was 101·5. That foal also got scour and died. I then got a third one with a temperature of 103, and that took scour and died. Where did they get the infection? Every possible precaution was taken, and the place was beautifully clean.

* Paper by Prof. A. E. Mettam, B.Sc., at the meeting of the National Veterinary Association at Windermere, Sept. 10th, (*ante*, pp. 293, 308 332.)

I had the scour break out in other studs, and my impression was precisely that of Prof. Mettam, that the infection took place through the umbilicus. I then took every possible precaution to avoid infection through the navel, and I must say I had a good deal of success. I am perfectly certain that in several cases where infection occurred it took place through the navel, and had strong suspicion that the source of the malady laid in the maternal passage. After that I thoroughly washed the mare with antiseptic before foaling. I have had several outbreaks of scour amongst calves, and I took every possible precaution in the places. I even took the cowmen away and got fresh men, and had the cows thoroughly washed and the navels dressed, but even then I got attacks of the scour. But almost invariably the navels began to sweat, so that my impression is that although I dressed them thoroughly well with antiseptic on the surface, the infection took place probably in transit through the vagina and the germs had penetrated deeply into the tissue.

Mr. A. Spreull: There is a very considerable trade in calves between the North of England and Forfarshire. Very often the calves come to us in a perfectly healthy condition, but in other cases we have outbreaks of scour amongst them. What these outbreaks of scour arise from I do not pretend to say, but they occur, and sometimes are pretty severe, a large proportion of the calves being lost through it. I do not think it is possible that they get contaminated on the way; I suspect it arises at the point from which they are sent off, probably from the introduction into the consignment of one or more diseased animals, which in transit communicate it to the others, and in that way propagate the disease. However, that aspect of the question can be more properly dealt with by those who are more familiar with the bacteriological part of the subject, and I leave it to them with a considerable amount of confidence. There is another experience of mine which I should like to relate in regard to an outbreak which occurred, not amongst calves that were conveyed from any distance, but amongst calves that were brought up on a particular farm where the disease was never known to have occurred before, and where, I may also say, it has never occurred since. During the spring that I refer to, the calves were sucking the mothers as usual, being confined in courts throughout the steading of a large farm, and there the disease broke out amongst them, and I was called in to see if I could find the cause. I am at a loss now, as I was at a loss at the time, to spot any particular cause. Amongst other things, I examined the milk of the cows. I put the milk very carefully into glass phials, took it away, and examined it microscopically, and found that the little globules of milk comported themselves very peculiarly. Instead of being uniformly distributed throughout the liguid they arranged themselves into groups or masses very much of the nature of zooglæa, and as long as this went on the calves continued to take the disease. In the course of a few weeks the season had advanced sufficiently for the cow to be turned out, and, like the experience of other gentlemen which has been related to you, the disease entirely disappeared without any medical or other treatment being adopted. We attributed a large proportion of the deaths among the calves imported from the North of England to derangement of the digestive system, arising from the long want on the journey, succeeded by too liberal an allowance of milk on their arrival, and there is no doubt that the very heavy mortality amongst them was in some measure due to that.

Mr. W. A. Byrne: My experience is not large, as I do not live in a cattle breeding county, and my opportunity of observing this disease has been comparatively small; but from the few cases where I had the opportunity of investigating, following the lines recommended by Prof. Nocard, I was forced to the conclusion that it was possible, and, in some instances, almost certain, that the virus, whatever it may be, could not possibly gain access to the animal through the umbilicus. There is no doubt that the virus is contained in the house in which the cattle calve, and I do not think anyone can contend that this virus cannot gain access through the umbilical cord. There is no doubt that in the County of Limerick, where there is an enormous mortality in calves, that attention to the recommendations made by Prof. Nocard and Mettam has resulted in a very substantial diminution of the death rate.

Mr. Roberts: I lived in this district about 11 years, and I saw a great deal during that time of white scour in calves. There are two or three conclusions at which I arrived at the time, the first being that the material, whatever it is that causes the disease, is contagious. I had quite a large number of instances in which the farms were perfectly clear of the disease; it had never been known there until people came to the market, bought some calves, and took them home to the farm. Those calves were very often placed in pens at the market, and, if healthy when they were placed in the pens, they very soon broke out with the disease when they got to their new home. Another thing I noticed about the calves was this, that the material that causes the disease is of a very irritating nature, or else the irritation is spread from the membrane of the mouth and nose to the skin, for almost invariably the skin round about the nose was quite bare, the hair all fell off and left the skin quite as bare as one's hand. This was often the case also about the anus; the skin surrounding the parts became excoriated, and all the hair fell off. I think it is reasonable to infer that if the noses of those calves were put into the milk pails, and other calves fed out of the same pails, they would be likely to contract the disease, as well as from the excrement of the calves. The point I make is that it is a contagious disease.

Mr. W. H. Bloye: I should like Professor Mettam in his reply to state whether he thinks the white scour which occurs within 2, 5, or 7 days is identical with the disease which occurs within 3 or 4 weeks of the birth.

Mr. James Laithwood: One gentleman who spoke some time ago appeared to be at a loss to know how the calves that had been transferred from other counties to his own got the disease. I think there is no doubt whatever that if he had traced one or more of those particular calves to the place of birth he would probably have found a number of dead calves—or probably none at all. It is undoubtedly a fact that when a farmer gets this disease upon his premises it pays him best to immediately sell the calves as soon as they are born, and as a rule he sends them straight away to the market. That is one method of transferring the disease from one farm to another, and spreading it all over the country more or less. Considering these facts, in my opinion this disease ought to be included in the Contagious Diseases of Animals Act, and any farmer with premises affected with this disease ought not to be allowed to sell any animals under a certain age from such premises for the purpose of transfer to others.

Mr. Joseph Hewson: I wish to give you my experience of this disease, which I am sorry to say is very prevalent. First of all I would like to point out that it appears very differently in different places. The disease is often seen a few hours after birth. In one outbreak with which I was concerned the calves were seized with the disease a few hours after birth, generally about 6 hours. As a result of the disease the calves invariably die; in the outbreak of which I am speaking I do not think one calf got better. The people gave up sending cows to the auction market to calve, which they had been in the habit of doing, and about six months afterwards there were no cows calving in that particular

place. After that time had expired the cow dealers began sending the cows back, and there was no further trouble with the complaint. The auction market was thoroughly disinfected; while the outbreak was on we washed it out, and even changed the place in which the calves were reared, but still the trouble persisted until the cows were stopped being sent there. I had no idea what was the cause of the trouble until the discussion this morning, when Mr. Hughes spoke about the probability of the infection being started by the manner in which the calves are first fed, *i.e.*, by teaching the calves to drink milk by sucking the fingers placed in the milk pail. That seems to me a very probable explanation of the way in which the disease is spread and the reason why it occurred so soon after birth in this outbreak in the auction house.

There is another thing which I should like to bring before the attention of the meeting which seems to me to be another form of the disease, namely, what we call navel-ill in calves. It is a disease which is very prevalent in the district in which I practice. It generally appears from 24 hours up to a week after birth. On examining the navel we find it enlarged, thick, and very much inflamed. Very often at the same time scour appears, but the funny thing about it is that it is so amenable to treatment when you would think that there was no chance whatever of the calf-getting better, and that it had only a matter of a few hours to live. The treatment I adopted was to lay the navel open and dress it with a strong antiseptic—I use ferric chloride—and that is all that is done with the exception of giving a strong dose of castor oil or some other purgative. Within 24 hours after that treatment the calf is practically well again. That is a very common form of disease in Cumberland. With regard to the disease in foals, that is a different disease altogether; it is different to white scour; there is no diarrhœa.

Mr. T. Eaton Jones: We have heard of two probable sources of infection in the navel, but I should like to mention a case showing that another source of infection may be probable. I cannot prove it, but I simply suggest that the infection may have taken place in utero through the partially dilated os. About 7 years ago a foal was born from a very good mare, and a month after birth it developed navel disease. The foal was treated for the disease and was cured, but unfortunately it always remained with very large hocks and knees and deformed fetlocks. But she was able to work on the land, and was put to the farm and able to plough. She grew to an immense size, about 17·2. When she was 3 years of age she was put to a horse, and the first foal that was born from her was born with the disease, with tremendously large hocks and knees, large fetlocks, and the joints twisted and generally out of shape. The foal was destroyed, and the mare was put to another horse. In the following year a foal was born of exactly the same kind. The mare was then put to another horse, and last year she dropped a very good foal, which was perfect in shape and everything else. On Monday last I had a message to go and see the foal, and found that she was undoubtedly suffering from a form of septicæmia. I could not find any cause for the septicæmia at all. The hocks and the knees were large and the breath was stinking. It really seemed to be a case of septic pneumonia more than anything else, and she died in about 5 or 6 hours. The post-mortem examination revealed that there was a general peritonitis, abscess in the liver, septic pneumonia, and other indications of general septicæmia. About a month or so afterwards the dam was destroyed and various abscesses were found, septic pneumonia and everything else. I have never heard the theory advanced before, but is it possible that the germ may have laid in the womb all those years or not, and is it possible that the disease may take place through the partially dilated os before the foal is born? I should like to know whether Prof. Mettam thinks that is possible.

Mr. G. A. Banham: When the author replies it would be of great interest if he could give us his opinion as to the possibilities of heredity of this disease. After what Mr. Jones has told us I think there is some reason for asking that question. Not only so but I can point personally to several mares which I could guarantee would throw foals with this disease. We have followed up the matter now for some five or six generations; but where the infection comes from I do not know. We have changed the horse over and over again, but there are certain mares who, as sure as they throw a foal, will throw a foal which will develop the disease within a week or a fortnight. We have changed the surrounding and done everything that can be done; the mares are in the best of stables, and are under the control of the most cleanly people that you could have. Sometimes the mares foal in, and sometimes out.

Mr. Kendall: I should like to ask Professor Mettam if there is any reason why scour in calves should stick to one particular farm rather than spread to other places equally ill-kept and dirty? I suppose that farms in this district are pretty badly kept, as far as the rearing of young stock is concerned. The usual method is to allow all excrements to collect right through the winter; it is moved once a year, in the spring; they cart it direct from the boxes to the field, and frequently they cut it with a hay knife. It is a notorious fact that certain farms are affected with scour and that no foals can be reared that are born indoors, There are other farms which are kept under similar conditions, and which even adjoin these infected farms, where they never lose a foal, and I should very much like to know if the author can give any reason why one particular place should be infected and another not?

Professor Mettam, in reply, said: I am extremely obliged to you for the very kind reception you have given to the paper. I am quite aware, and no one more so, how faulty it is. But, nevertheless, I rejoice that we have had a very good discussion. I should like, if I may say so, to express the extreme pleasure it is to me that my old teacher, Prof. M'Fadyean, should have been the one chosen to open the discussion. Prof. M'Fadyean has asked me several questions on points which I have not been sufficiently explicit about, and he tells me that his opinion of septicæmia and mine, as expressed in the definition, are not one and the same. I quite agree that the definition of septicæmia, as it is laid down there, might have been extended, but the lesions that we saw in our very acute cases of white scour, more especially in our experimental cases, undoubtedly were of true septicæmic type, and I am quite convinced that if he had seen some of the cases that I saw, he would at once, in one word, have summoned up the lesions as septicæmic. I will tell you one most prominent case. It was that of an experimental animal, on which we were making a post-mortem one Sunday afternoon. As soon as we opened the carcase—I may say that we did not expect the animal to die, although it had had a large dose of the virus—I remember Professor Nocard looking into the carcase through the incision in the abdomen and say, "Charbon?" He apparently, on a first glance at the carcase, thought it was a case of anthrax, and he satisfied himself that it was not by going to a laboratory and staining a blood film. The term I put down there was merely to explain myself in regard to the lesions which we commonly see in the most virulent type of what I term here white scour. But, then, I come to the conclusion from the discussion that has gone on that white scour is probably a mixed infection. I am bound to say that the cause of white scour in every instance is not the same. Of course, we must admit that the term white scour, when all is said and done, is only a symptom of a disease. It is only diar-

rhœa. It is diarrhœa of a particular disease, a diarrhœa probably that is associated with the food that the animal is getting. I, myself, have very little doubt in my own mind that the organism which Nocard discovered, and which Lesage and Delmer, working independently, found simultaneously, does produce a disease or lesion in young calves which the laymen, and which we call white scour. As I say, the term, "white scour," is only a conventional term and one applied to the symptom of the disease. That also partly replies to the question that my friend, Prof. M'Fadyean, put to me as to why we did not use ingestion, by using the dejecta of diseased calves and feeding two calves. That was one explanation, because, of course, I need not remind the audience that the intestinal flora is rich in micro-organisms of all sorts. A number of organisms have been blamed as producing white scour, and I think Prof. M'Fadyean will agree with me that many of these organisms are probably derivatives or allied organisms belonging to the colon group. Jensen, whose experiments I mentioned in my paper, certainly produced the disease by giving animals milk in which the dejections were mixed; but when we have a number of micro-organisms, warring, as it were, in the alimentary tract, it is quite possible that some of them may lower the vitality of the individual and thus allow the entrance of organisms into the body which produce a toxin which produces death. In regard to the question of prophylaxis, to which attention was drawn, where I had provided two strings to my bow, I wanted first to expresss my belief that, supposing careful attention is paid to the umbilicus—I mean attention as one of ourselves would conscientiously give in a case where we have a good opportunity—and if the navel is disinfected immediately the animal is born, in my judgment, this thing which we call white scour is not likely to infect that animal: but, in order to have two strings to one's bow, as it were, there is no reason why we should not insist upon thorough cleanliness in places where young animals are kept; indeed, in other words, we might consider that thorough cleanliness is a capital addendum to any treatment that we care to adopt.

Mr. McGavin in his remarks gave what I must consider as being a considerable amount of support to certain of my contentions. He showed that this infection ran, as it were, in certain houses. That also we found, and we also found, as Mr. McGavin found, that when the animals were turned out to calve, and were removed from the house in which the infection lay, that those cows, in a great majority of instances, produced calves that lived, which did not suffer from white scour at all. His experience of amateur treatment is the experience of those who were engaged in the white scour investigations. You cannot trust the peasant or the farmer's man to carry out accurately and fully your recommendations. There is some one thing or other which they will not think necessary to do, and it is by allowing a hiatus of that description in the treatment that very frequently the infection creeps in despite yourself. In the early days of the white scour investigation I instructed a farmer, a very intelligent man, to carry out these recommendations as we would carry them out ourselves. I went to a considerable amount of trouble in explaining even to the most minute detail, how very necessary it was that the antiseptic should be rigidly employed. I obtained a disciple, and one who stuck to his text, and that man to my knowledge treated 40 calves on different farms where white scour was practically annihilating the calves before the treatment came in and saved 39 out of 40, the only treatment that was adopted in these instances being the umbilical treatment. The white scour which I am referring to is that disease from which young animals die during the first day of birth, or in the few days succeeding birth, or that die within a week. Mr. Cameron asked a question as to the purchase of calves, and the bringing of them into byres or shippons and stated that these calves brought in the disease, or that calves died in these byres or shippons after they have been brought into contact with animals brought from a distance. I was very glad that Mr. Cameron referred to that point. He also wanted to know the source of infection. I will tell you the reason why I am glad he asked me about that. I know a particular farm; it is not more than a mile or two from the town where Mr. Howard practises, in which for years they never had a case of white scour upon it, although white scour was prevalent all round, the calves being practically annihilated. On this particular farm 70 cows were kept, and, as a rule, 70 calves were born. But it happened in one year that they had more milk than they could use up with the calves they had, and the owner told me he went into market and purchased calves. He purchased in the open market calves that were sent into the market by men who were in the position of the clients of some of the gentlemen who spoke here this morning—they got rid of their calves as soon as they could because they were afraid of their dying on their hands. The peasants and the farmers in County Limerick know exactly what will happen if they keep their calves, so they prefer to let them die on somebody else's hands rather than their own. This man went into Limerick market, purchased calves, took them back to his farm, and they commenced to die from white scour, and from that time up to now he has been in the habit of losing calves, until no less than 90 per cent. of the calves that were born upon his farm died. In that case, in my judgment, the disease was brought on to the farm by calves that were infected with the disease when purchased in the open market. I was very pleased to hear what Mr. James had to say. I also have seen calves dying from white scour when they have been sucking their mothers; I also have seen calves dying from white scour when they have had muzzles on them. The milk that was given to them had been taken into the house and brought out of the house after a short time and fed to the calves. In certain instances it has been taken straight away from the cow to the calves, and during the interval the calves themselves had been muzzled. I cannot possibly see how the calf could have taken infected material (unless the milk had been infected in the first instance) into its alimentary tract, when it is muzzled with a wicker basket. Mr. Pugh recalls some very interesting cases where he undoubtedly had moist gangrene of the umbilicus, and it is probable that he had in in most cases an umbilical infection. Mr. Roberts' experience supports what several of the speakers said. Then Mr. Bloye asked the question, Is the white scour in the early days of extra-uterine life the same as the white scour of 3 weeks? I do not believe it is at all. Mr Laithwood also referred to the same point as Mr. Cameron and Mr. Roberts. Mr. Hewson mentioned very interesting cases of calves dying a few hours after birth. That also was a problem which I suggested to Prof. Nocard that he should investigate, as to whether there was any such thing as intra uterine infection. We purchased some cows, very fine cows they were too, obtained from a part of Ireland where the white scour that we were investigating was unknown. One cow received into a jugular vein 5 c.c. of a culture about 11 o'clock on a Saturday morning, and at half-past 1 or 2 o'clock on the Sunday afternoon she died. As it turned out she was pregnant. We took special precautions to take out the uterus intact and to examine on the most approved lines the blood of the unborn calf. Extensive cultivations were made upon different media, but in no instance was there any sign of infection in that calf. Evidently, as Prof. Nocard put it in his report, the placental filter was a perfect filter as regards this particular infection.

Then Mr. Jones and Mr. Banham ask me whether this disease is hereditary. I do not think so at all. I thank you very much indeed for the honour which you paid me in asking me to prepare this paper, and again I say how agreeably pleased I am that my dear friend M'Fadyean opened the discussion. (Cheers.)

SLEEPING SICKNESS IN UGANDA.

The Discovery of the Cause.

Lieut.-Colonel David Bruce, R.A.M.C., F.R.S., returned to England from Uganda on September 28, and has brought with him a report containing some very important observations on the etiology of sleeping sickness. Some conception of the ravages wrought by this disease among the native population in Uganda may be formed from the condition of the Buvuma Island, where a population of 22,000 has been reduced to 8,000 since the outbreak of the disease, while the provinces of Busoga has been in the southern parts almost depopulated.

Colonel Bruce was despatched by His Majesty's Government, acting on the advice of the Royal Society, to superintend a further investigation into this disease, upon which some work had already been done by the previous commission sent out by the Royal Society. He was accompanied by Dr. Nabarro as bacteriologist, and, while, in Uganda, was joined by Captain Grieg, I.M.S., who had been sent out by the Indian Government for the purpose of studying the disease.

Colonel Bruce and his party reached Entebbe on March 16, and, on arrival, found that Dr. Castellani, a member of the previous commission who had remained at work upon the investigation, had observed in five cases of sleeping sickness trypanosomes in the cerebro-spinal fluid, and, in one case, in the blood. At that time Dr. Castellani did not consider that this parasite had any causal relationship to the disease, but thought it was an accidental concomitant. Nevertheless, the observation was of the greatest importance to the further investigations; and, as soon as it was reported to Col. Bruce, whose well-known investigations into the tsetse fly disease in South Africa had given him a special familiarity with this genus of parasite, the whole party, with the most valuable and willing assistance of Dr. Moffat, the principal medical officer, and his assistant medical officer, addressed themselves with ardour to the pursuit of this line of investigation; with the result that, by the first week in April, the date of Dr. Castellani's departure from Uganda for England, out of 34 cases of the sickness examined, 70 per cent. were found to have this parasite in the cerebro-spinal fluid, whereas control cases of patients under treatment for other diseases in the native hospital, when examined in the same way, showed no trypanosomes in their cerebro-spinal fluid. The further progress of the investigation increased this proportion to 100 per cent. of the cases examined, in all of which the parasite was now found not only in the cerebro-spinal fluid, but also in the circulating blood; pointing to the very strong probability (as telegraphed to the Royal Society by Colonel Bruce at the beginning of April) that this trypanosoma was the cause of the disease. A series of carefully controlled and minutely observed experiments was carried out by Bruce, Nabarro, and Grieg, in the course of which it was discovered that monkeys inoculated with cerebro-spinal fluid from sleeping sickness patients, or with blood from natives not as yet showing symptoms of sleeping sickness, but containing a similar parasite, sickened and died with all the symptoms of sleeping sickness.

From the analogy of the closely related disease in cattle, the nagana or tsetse fly disease of South Africa, it was suspected that in sleeping sickness a like method of infection took place. Along the lake-shore numbers of a tsetse fly (*Glossina palpalis*) were found, and it was demonstrated by experiment that not only were these flies, fed on sleeping sickness cases, capable of conveying the disease to healthy monkeys, but that the freshly caught flies from an infected area, without any artificial feeding, were also capable of conveying the disease.

It was further discovered by a carefully-organised investigation, in which the native Regents of the Protectorate rendered valuable assistance, that this fly, like its congener the tsetse fly of South Africa, is confined to well-defined areas, and that these areas correspond absolutely with the distribution of sleeping sickness; whereas, in regions where no *Glossina palpalis* is found, although other biting flies abound, there is no sleeping sickness. Moreover, an examination of a large number of individuals in the sleeping sickness areas and the non-sleeping sickness areas respectively revealed the fact that, while a large percentage of the inhabitants of the sleeping sickness areas have in their blood the trypanosoma already referred to, in not a single case taken from inhabitants of non-sleeping sickness areas was this parasite found.

These investigations, therefore, point to the conclusion that sleeping sickness in caused by the entrance into the blood, and thence into the cerebro-spinal fluid, of a species of trypanosoma (probably the *Trypansoma Gambiense* discovered by Forde and described by Dutton) which is transmitted from the sick to the healthy by a species of tsetse fly (*Glossina palpalis*) and by it alone; that, in short, sleeping sickness is a human tsetse fly disease.

Nothing is more curious, in the page of natural history which this discovery unfolds, than the differences which exist among animals of different species, and among men of different races, in their liability to suffer from this or that fly communicated disease. No case of sleeping sickness has as yet been observed in a European, although many Europeans have lived in the infected countries, and have probably been bitten by the *Glossina palpalis* over and over again. The bite of the tsetse fly, although rapidly fatal to horses, appears to be altogether innocuous to human beings, whether black or white; and the negro race in malarial districts acquires something approaching to complete immunity from that disease. The blood of the native children swarms with the malarial parasite, and thus, although not suffering in health themselves, they are the main sources of supply to the anopheles of their localities, so that the vicinity of native quarters becomes a great source of danger to Europeans. In the case of malaria it was discovered, long before the parasite was known or suspected, that the fever might be cured by quinine; and the discovery of the parasite led to the further discovery that quinine acted upon it as a poison, and that, to be given with the best effect, it must be so timed as to be present in the blood at a particular stage of the parasitical development. It must, of course, be hoped that the trypanosoma may admit of being killed in the blood in some similar manner; but the lethal agent has first to be found. It is reported that three natives suffering from sleeping sickness, which is often a long-continued, although always a fatal, malady, have been brought to Paris for the purpose of observing their symptoms; and there can be no doubt, if this be so, that the search for what must, we suppose, be styled a trypanosomacide will be actively conducted, of course with all due regard to the condition of the patients themselves. It is evident that we are as yet only upon the threshold of a knowledge of the manner in which the welfare of mankind may be affected through the medium of insect life of various kinds.

To the Editor of *The Times*.

Sir,—In *The Times* on Saturday, November 7, you published a short summary of the result obtained by the inquiry into sleeping sickness carried out under the direction of a committee of the Royal Society. In the same issue of *The Times* there appeared a leading article on the subject. The result reported on is briefly that sleeping sickness is due to a trypanosoma parasite similar to that of the notorious tsetse-fly disease and carried from man to man by a fly—*Glossina palpalis*—similar to, though not identical with, the South African tsetse fly, which carries the nagana-trypanosoma from big game animals to domesticated horses and cattle.

There are some inaccuracies in the leading article of Nov. 7th, which suggest an erroneous view as to the merit of the various persons concerned in this investigation. I have a special knowledge of the various steps of this inquiry, since I have served for seven years on the "Tsetse Fly Committee" and on the "Malaria Committee" of the Royal Society, which undertook, at the request of the Government, the investigation of sleeping sickness in Uganda. I trust, therefore, that you will allow me to correct the misapprehensions caused by some of the statements made in your leading article.

It is of serious public importance that in such matters as this honour should be given where honour is due, and that no mistake on such a matter should obtain currency through your authority.

In the first place, in 1902, three young medical men, Doctors Low, Christy, and Castellani, were sent by the Royal Society Committee to Uganda to study the disease. After some months Dr. Castellani reported that he had discovered a bacterium (streptococcus) which he definitely announced to be the cause of the disease. The other two observers were unable to come to any conclusion as to the cause, and returned home with information of more or less value as to the distribution and features of the disease. Dr. Castellani alone stayed on in Uganda, in order to work out more fully the history and nature of the bacterium which he held to be the cause of the disease.

Your article says that "some" of the members of this first expedition "returned to England without being able to present a report which was conclusive, although more than a suspicion of the truth had suggested itself to their minds."

This statement, I have no hesitation in saying is absolutely incorrect. Neither Dr. Low nor Dr. Christy had the vaguest suspicion of the truth; nor was there at the time of their return any fact known to justify a suspicion of the connection of trypanosoma with sleeping sickness. Neither of these gentlemen has, either before or after the great discoveries, stated that he had any suspicion of the truth.

Some time after the return of Doctors Low and Christy, the Royal Society Committee, finding that the inquiry had made no satisfactory progress, determined to advise the Foreign Office to secure, if possible, the services of an observer of experience and proved skill—namely, Colonel David Bruce, F.R.S., of the Army Medical Service, the discoverer of the blood parasite in the nagana or tsetse-fly disease of South Africa.

I may say, in parenthesis, that the committee had not been able, in the first instance, to despatch an investigator who had already proved his capacity in this difficult field of discovery, owing to the fact that they had but small pecuniary resources at their disposal.

Colonel Bruce, accompanied by Dr. Nabarro, reached Entebbe on March 16th. There he was met by Major Greig of the Indian Medical Service. *The Times* leader says that Dr. Castellani, on the arrival of Colonel Bruce and his party, was "able to submit to them evidence of a small protozoa (*sic*) known as a trypanosoma in the blood and spinal fluids of the affected persons." A misapprehension is likely to arise from this statement, and indeed has arisen, as I will show below. The suggestion is that Dr. Castellani submitted this parasite, holding it to be the cause of the disease. Full credit is due to Dr. Castellani for his discovery of the presence of the trypanosoma, and has been given to him in no unmeasured terms by Colonel Bruce in his official report to the Royal Society's Committee. But it is neither more nor less than the fact that Dr. Castellani came upon this trypanosoma on five occasions only in the cerebro-spinal fluid and once in the blood of sleeping-sickness patients, when looking for the streptococcus which he held to be the cause of the disease, and that he informed the head of the medical service of Uganda on the very morning of Colonel Bruce's arrival in Entebbe that he regarded the trypanosoma merely as an accidental concomitant of the disease and as having no causal relationship to it. Dr. Castellani's attitude in this matter is stated in the official summary published in your columns at the same time as your leading article. On his return to England, which followed shortly after Colonel Bruce's arrival in Entebbe, Dr. Castellani maintained in an official report to the Royal Society Committee (published some months ago) that the streptococcus to which he had originally committed himself was, though accompanied by the trypanosoma, an effective agent in the production of sleeping sickness.

In your leading article there is no mention of the departure of Dr. Castellani from Uganda. The suggestion is that he stayed and completed the further investigation by which it was discovered that the new parasite (trypanosoma) is present in all cases of sleeping sickness and is communicated from the sick to the healthy by the bite of the fly *Glossina palpalis*.

The truth is that the importance of the occurrence of a trypanosoma in a few cases of sleeping sickness was apprehended by Colonel Bruce, and was not perceived by Dr. Castellam. It was Colonel Bruce who immediately organised a thorough and successful search for the trypanosoma in all cases, and who, after Dr. Castellani's departure, by an energetic system of collecting the flies present in some hundred different localities around Entebbe, showed, firstly, that the fly *Glossina palpalis* is abundant, though partially distributed in that region; and, secondly, that where it occurs sleeping sickness occurs, and where it is absent sleeping sickness is absent. This was followed by direct experiments on inoculation by the fly.

That a misapprehension has arisen as to the real credit due to Colonel Bruce in this matter is evident from a leading article published two days after your own in a Scotch newspaper (*The Aberdeen Free Press*), in which it is stated that a medical commission was sent out some years ago to study sleeping sickness in Uganda, and that one member of the commission, Dr. Castellani, "has been at work on the spot ever since, and to him, as appears from a communication to *The Times*, belongs much of the credit for the discovery of the cause of sleeping sickness, the achievement of which marks a distinct triumph for medical science." The facts that Dr. Castellani had nothing to do with the later investigations, the demonstration of the invariable presence of the trypanosoma and the existence and concomitant distribution of the fly *Glossina papalis*, are not mentioned, but, on the contrary, his participation in that work is implied. I should regret extremely were it to be supposed that I wish to detract from the merit which really belongs to Dr. Castellani. An investigator is always entitled to full credit for the discovery of a fact which proves to be one of great importance, even though he may not have fully appreciated that importance. Dr. Castellani discovered the presence of a

trypanosoma in some cases of sleeping sickness; that is his merit. Colonel Bruce would not have been able at once to pursue his investigations if Dr. Castellani had not ascertained the initial fact before the former arrived in Uganda. Possibly Colonel Bruce would have himself come upon the trypanosoma after a few days' work in Entebbe. In any case, it is greatly to be desired that the share of honour proper to each discoverer should be given with an approximation to justice, and for this reason I venture to ask you to publish this letter.—Yours faithfully,

E. RAY LANKESTER.

Natural History Museum.
Nov. 16.

THE AGRICULTURE OF LONDON.

Ten years have elapsed since the agricultural returns for the County of London—an area carved out of the counties of Middlesex, Surrey and Kent—were first officially collected. An inquiry into the changes which have since occurred brings to light one unvarying story of decline, as regards both the acreage of crops and the number of live stock. The County of London, as defined in the Local Government Act, covers an area of 75,442 acres, of which 31,484 acres are in Middlesex. 23,898 acres are in Surrey, and 20,060 acres in Kent. It comprises the Parliamentary boroughs of Battersea and Clapham, Bethnal-green, Camberwell, Chelsea, Deptford, Finsbury, Fulham, Greenwich, Hackney, Hammersmith, Hampstead, Islington, Kensington, Lambeth, Lewisham, City of London, Marylebone, Newington, Paddington, St. George (Hanover-square), St. Pancras, Shoreditch, Southwark, Strand, Tower Hamlets, Wandsworth, Westminster, and Woolwich. The total cultivated area has diminished during the decade from 14,927 acres to 10,163 acres, the shrinkage of 4,764 acres amounting to one-third, and representing an average loss of 476 acres per annum. In 1893, nearly one fifth of the entire county area was under agricultural cropping; this year the proportion is less than one-seventh. In the subjoined table the figures for 1903 are compared with those for 1893:—

AREA UNDER CROPS AND NUMBER OF LIVE STOCK IN THE COUNTY OF LONDON IN 1893 AND 1903.

CROPS.	1893	1903	Decrease in 1903.
Corn crops ... ... Acres	812	334	478
Green crops	2,840	1,815	1,025
Clover, sainfoin, and rotation grass	401	128	273
Permanent grass	10,182	7,544	2,638
Small fruit	332	244	88
Bare fallow	360	98	262
Total cultivated land	14,927	10,163	4,764
LIVE STOCK.	No.	No.	No.
Horses	991	718	273
Cattle	7,645	4,940	2,705
Sheep	5,438	3,703	1,735
Pigs	3,233	2,229	1,004

The extent of land under corn crops has diminished considerably more than one-half, and at 334 acres it represents an area less than that devoted to corn on many an Eastern Counties farm. There is not now 100 acres of any single kind of corn, the total of 334 acres comprising 99 acres of oats, 96 of wheat, 52 of rye, 36 of barley, 30 of peas, and 21 of beans. Green crops have sacrificed more than one-third of their acreage of ten years ago, and are exactly 100 acres less than last year. They include 351 acres of potatoes, 282 of mangel, 267 of cabbage and rape, 92 of vetches or tares. 21 of turnips, and 802 acres of "others,"—doubtless market garden crops. Lucerne is returned as occupying 57 acres. Temporary grass land, comprising clover, sainfoin, and grasses grown in rotation, has declined seven-tenths on the decade, and now stands at no more than 128 acres. Permanent grass—that is, grass land not broken up in rotation—has diminished by more than one-fourth, its present area being 7,544 acres. Three-fourths of the agricultural land of the county of London is thus in permanent pasture, but this has been shrinking at the average rate af 264 acres per annum during the last ten years. The great London parks are included in the area of permanent pasture. Of this year's total of 7,672 acres of grass lands of all kinds, 3,300 acres, or less than one-half, were mown for hay. The extent of land occupied by small fruit has decreased by one-fourth to 244 acres. The area of grass land under orchards this year is 182 acres, which differs but little from the 190 acres of ten years ago. The aggregate loss of 4,764 acres —or about 7½ square miles—of agricultural land in the space of ten years tells clearly enough of the rate at which the farmer is being driven out of the metropolitan area.

Horses have diminished by more than one-fourth, the present total of 718 being made up of 621 horses used for agricultural purposes (including mares kept for breeding), and 97 unbroken horses. The total cattle have declined nearly one-third on the decade, and of this year's number of 4,940 as many as 4,689 are specified as cows and heifers in milk or in calf. The following statement shows the numbers of these milking cows in each of the last 11 years:—

6,657 in 1893	5,648 in 1897	4,972 in 1901
6,173 in 1894	5,802 in 1898	4,669 in 1902
5,686 in 1895	5,467 in 1899	4,689 in 1903
5,825 in 1896	5,040 in 1900	

From a total of 6,657 ten years ago the milking cows of the metropolis have declined to 4,689, a loss of nearly one-third. It is more than probable that in the course of time the urban cowsheds with their occupants will disappear, and the metropolis will become entirely dependent upon external sources for its daily supplies of milk. Not much interest attaches to the numbers of sheep, as a few more less awaiting the butcher's knife on the day of the census would have a considerable proportionate effect upon the total. It is seen, however, that on the decade there is a decline of nearly one-third. Of this year's total of 3,703, only 289 were ewes kept for breeding, whilst 339 were lambs. Pigs, like sheep, have diminished nearly one-third. Out of the current total of 2,229 not more than 256 were returned as sows kept for breeding.—*The Times.*

The Tuberculin Test.

The Cape authorities have put their foot down in the matter of disease. Somebody has been kind enough to send a diseased animal to the Cape. Forthwith all stock to be sent there must be certified to have undergone the test. Whatever the Briton may think, his customers abroad seem quite determined to stand by tuberculin. Would it not be sound policy for the British stockowner to recognise the situation, and pay some serious attention to the test? Mr. Taylor, in his extremely useful lecture at East Kilbride, referred to the test, and made it plain that he, for one, believes in it when properly applied. A practical V.S. like Mr. Taylor has many opportunities for finding out whether this view is sound. Mr. Chamberlain, in his great crusade, asks whether it is conceivable that all the world is wrong and only Great Britain right in this matter of fiscal policy. Is it conceivable that all Britain's customers for pure-bred stock are wrong, and only British stockowners right about tuberculin? Until the latter make a serious effort to test the test, they are likely to find themselves in a minority on the point.—*The Scottish Farmer.*

DISEASES OF ANIMALS ACTS 1894 & 1896.

SUMMARY OF RETURNS, WEEK ENDING NOVEMBER 28, 1903.

Period.		Anthrax.		Foot-and-Mouth Disease.		Glanders (including Farcy)		Rabies. Cases Confirmed.		Swine Fever.	
		Out-breaks	Ani-mals.	Out-breaks	Ani-mals.	Out-breaks	Ani-mals.	Dogs.	Other	Out-breaks.	Slaughtered *
Week ended November 28, 1903 ..		16	28	..	..	19	43	..	..	28	136
Corresponding week in	1902 ..	11	14	..	..	19	30	..	..	32	146
	1901 ..	23	31	..	..	26	43	..	..	25	105
	1900 ..	15	21	..	..	16	33	..	1	27	201
Total for 48 weeks, 1903		695	1041	..	..	1364	2325	..	..	1379	7302
Corresponding period in	1902 ..	622	964	1	120	1078	1927	12	11	1583	7687
	1901 ..	585	847	12	669	1270	2198	1	1	3036	14696
	1900 ..	511	856	17	227	1046	1740	6	5	1801	16804

NOTE.—The figures for the Current Year are approximate only. * As Diseased or Exposed to Infection.
Board of Agriculture and Fisheries, Dec. 1st, 1903.

THE LIVERPOOL UNIVERSITY VETERINARY MEDICAL SOCIETY.

Veterinary Research Laboratory,
University College, Liverpool.

Dear Sir,

After your Editorial in last Saturday's *Record* perhaps the following may be of interest.—Yours faithfully,

R. E. MONTGOMERY.

By permission of the Council of the University of Liverpool a conversazione was held on Thursday, Nov. 26th in the Veterinary Research Laboratory of that University. Amongst those present were Professor Boyce, F.R.S., Major Ross, C.B., F.R.S., Professor of Tropical Diseases, Professor Rushton Parker, Dr. Annett, Dr. Grunbaum, Director of the Cancer Research, and about 90 members of the veterinary and medical professions from the adjoining counties and many others interested in the movement.

In the laboratory an exceedingly interesting collection of both microscopic and macroscopic specimens of all the contagious diseases of animals and the various neoplasms were on view. Lantern slides to the number of nearly 200 illustrative of the various pathological processes, parasites, bacteria etc., were so arranged as to be readily seen. Cultures of the pathogenic bacteria were also in evidence, as well as those found in air, soil and water.

Certainly the most interesting exhibit of all was the collection in both stained and fresh specimens of nearly every known trypanosome found in either man or animals, arranged for microscopical examination. As this is the most complete collection of trypanosomes to be found in any institution in the country it may be well to enumerate them : surra, nagana, Lewisi of rats, mal de caderas, dourine, gambian horse and T. gambiense (Dutton), these two latter having been discovered by Drs. Dutton and Todd of this school.

After tea a tour of inspection was made through the various laboratories affiliated with that of veterinary research, namely the two Thompson-Yates laboratories for pathology and physiology, and the three endowed together with that of veterinary research by Mr. Johnston, those for the Liverpool School of Tropical Medicine, for cancer research and for bio-chemistry. As all these laboratories are in direct communication it can readily be understood the work of each is greatly facilitated.

A meeting was subsequently held under the Presidency of Prof. Boyce to consider the advisability of forming a Veterinary Medical Society in connection with the University.

It was proposed by Mr. H. Sumner (Liverpool) seconded by Mr. W. J. Fletcher (Wrexham), supported by Messrs. J. B. Wolstenholme (Manchester) and Wm. Woods (Wigan), and carried unanimously that such a society be formed.

After due consideration the following scheme was decided upon.

Name proposed : The Liverpool University Veterinary Medical Society.

Membership : To be open to all members of the veterinary and medical professions.

Subscription : £1 1s. per annum, to include the following :—

Objects :—

1. To promote the study of Veterinary Science in all its branches.
2. To afford opportunity for the regular holding of meetings at which subjects of veterinary interest may be discussed.
3. To form a Veterinary Bureau to which members may refer for information on matters relating to their practice.
4. To form a Museum where specimens of interest may be collected, preserved, identified, examined, and catalogued.
5. To establish a Library of current literature and works of reference in a room set apart for this purpose.
6. To carry out microscopic examinations of morbid material of every description free of charge to members and to report thereon ; and to assist in the investigation of occult diseases.

The office bearers were then elected as follows :

On the motion of Mr. Eaton Jones (Liverpool), seconded by Mr. J. McKinna (Huddersfield), Professor Boyce was unanimously elected President for the ensuing year.

Vice-Presidents.— Messrs. H. Sumner, Liverpool ; Wm. Woods, Wigan ; J. B. Wolstenholme, Manchester ; and Dr. Annett.

Committee.—Messrs. J. McKinna, Stafford Jackson, W. J. Fletcher, J. Sumner, G. H. Locke, Eaton Jones, F. A. Ball, and H. H. Aldred.

Hon. Sec. & Treas.—Mr. R. E. Montgomery.

The following gentlemen, in addition to those enumerated above, joined the new Society.—Messrs. Heyes, Wood, Clarkson, Morgan, Walker, Barron, Noar, McLaren, Holburn, Berry, Richardson, Maquire, Scotson, Dixon, Adamson, Bate, Brooksbanks, Rogers, Dixon, Dr. Roberts, Prof. Rushton Parker, F.R.C.S., Messrs. Duggle, Edwards, Price, Lomas, and Dr. Ryder, D.V.S.

LINCOLNSHIRE VETERINARY MEDICAL ASSOCIATION.

The autumn meeting was held at the Albion Hotel, Lincoln, on the 15th ult., when Mr. H. C. Reeks, of Spalding (the President) was in the chair. There were present Messrs. T. B. Bindloss, Long Sutton; J. W. Gresswell, Peterboro'; R. W. Knowles, Wisbech; W. W. Lang, Caistor; T. W. Turner, Sleaford, hon. sec.

Letters of apology for non-attendance were received from Profs. Pritchard and Penberthy, and Messrs. Wragg, Mason, Nunn, Howse, Poles, J. Smith, Rudkin, Clarke, and Mackinder.

A letter was read from the V.M.A. of Ireland in reference to the exemption of veterinary surgeons to serve on juries, and a copy of *The Dublin Daily Express* wherein was a case tried before seven judges, which terminated in favour of the profession.

Mr. GRESSWELL contended that veterinary surgeons were equally exempt as those who prescribed medicine for the human subject, they were both medical practitioners. In Ireland he believed no veterinary surgeons were ever put on the jury list, and he did not think they were liable to be put on in England. He had mentioned the matter to the overseer of his parish, and that gentleman agreed with him that veterinary surgeons were medical practitioners, and although he said he should be liable to a heavy fine if he was wrong, he decided to keep the names of veterinary surgeons off the jury list, and he had not been on since.

Mr. BINDLOSS did not believe that veterinary practitioners were liable to be put on the jury list, but it seemed as if some of the overseers could not be made to understand this. He had appealed against his name being put on, but it was of no use, and he had received a summons to appear on the following Thursday. He had been told that he was not a medical practitioner.

Mr. TURNER remarked that they had to pass in veterinary medicine and surgery and be Registered according to the Act.

Mr. BINDLOSS said he had tried to explain that veterinary surgeons practiced medicine as much as the human surgeon, but they would not listen to him.

Mr. GRESSWELL did not think it mattered whether they gave medicine to the human subject or to animals. The Act said medical practitioners, and both of them practiced medicine. He was of opinion that the overseers had no power to put their names on the jury lists at all.

DISTRICT INSPECTORS UNDER BOARD OF AGRICULTURE.

The SECRETARY reported that he had had the resolution passed at the last meeting printed, and a copy sent to each of the Associations. Some of the Associations had written in approval of the resolution.

Mr. GRESSWELL said he could not see why qualified local men should have the police and distant men put in front of them. The local man got his living in the neighbourhood and knew the animals on most of the premises, and he was much less likely to make a mistake than a policeman or an inspector from a distance.

Mr. BINDLOSS asked if there were any of the copies of the resolution left, and on being informed there were, remarked that it would be a good thing if the members would send a copy to their own Members of Parliament.

The CHAIRMAN said if this was done some of the Members of Parliament would get several copies.

Mr. BINDLOSS thought this was a good thing, for the receiving of more than one would help to fix the matter more firmly in the member's mind.

It was then agreed that members of the Association who wished should be supplied with copies for this purpose.

SWINE FEVER.

By Mr. T. B. BINDLOSS, M.R.C.V.S., Long Sutton.

I notice that the objects of this Association are to cultivate and promote veterinary medicine and surgery, the advancement of the interests of the profession and the protection of their pecuniary and professional interests. There are different ways of aiming at these several objects and, thinking that it is not always the most scientific papers that do most to promote the interests of the profession, and being pressed at our last meeting by our President, I consented to introduce a subject for discussion.

You will remember that at Peterboro' a resolution was proposed, discussed and passed, and sent to the Board of Agriculture and also to the different Veterinary Medical Associations, disagreeing with the present methods adopted in the attempt to stamp out swine fever, and it must be pleasing to all members of this Association to know that we voiced what has proved to be the general opinion of the profession. This had something to do with my bringing before you this paper to-day and, though I do not attempt to bring before you anything new or very scientific, I hope that it will bring forth such a discussion that we shall all here agree as to what should be diagnosed as swine fever and what should not, and that, as far as we are concerned, there will be no contradictory diagnosis made in respect to this disease. If this object is attained then, in a small way, we must agree that something has been done in the advancement of the interest of the profession.

Swine fever is a disease presenting itself in many different ways and having a variety of symptoms. It is a disease that has been recognised as a specific contagious disease for nearly 40 years. In 1875 Professor Axe described it as being the exact counterpart of typhoid fever in man. In 1865 Dr. Budd, of Clifton, read a paper on swine fever under the name of pig typhoid.

It has been described under many different titles, among the commonest of which are: pig typhoid, hog cholera, red soldier, swine plague or schweine süche, infectious pneumo-enteritis, purples, etc. This last named synonym—purples—I think should be discontinued, as the disease commonly called purples in our district is not swine fever, and has no relationship to it—it is swine erysipelas.

Swine fever is caused by a bacillus known as the bacillus of swine fever. It is a short rod-shaped organism with rounded ends from 1-2 m.m. long, and ·6 m.m. broad, and thus is only about half the size of the tubercle or the swine erysipelas bacilli. It is non-sporulating, ærobic, and does not form filaments. It stains readily by the aqueous solutions of the ordinary basic aniline dyes, but not by Gram's method of staining.

The pig is the only animal naturally affected, and it is more commonly found among store pigs than among suckers or fat pigs. I consider that this is due not so much to a peculiarity of the working of the organism as to the fact that store pigs are taken from place to place by the dealers and are thus much more liable to come in contact with infected material. The common method of infection is by way of the mouth, the bacillus being introduced by ingestion, and certainly the common seat of lesions is the alimentary canal.

Symptoms.—These vary very considerably, and it is impossible to set down a table of symptoms that we can say are diagnostic of swine fever, for very often only one or two symptoms show themselves. I have had cases that on post-mortem examination have shown extensive lesions and yet, when alive, merely went off their feed for a few days. At the same time it is the rule that pigs that are suffering from swine fever have a very un-

thrifty appearance. As with all diseases of swine, the commonest symptom is the comparative loss of appetite, though in this disease there is not so much loss of appetite as capricious appetite. The animals will frequently approach the trough and eat but a very small quantity and then lie down again, and there is a general preference shown for sloppy foods or fluids. There is inclination to hide in the bedding, which is nothing more than a symptom of fever and a natural instinct on the part of the pig to keep its general surface warm. The temperature in swine fever does not reach that height that it does in some other pig diseases, but it will generally range from 104° to 106° in pigs that have been recently infected and the disease in the more active stage. This one might expect to find, considering that the disease takes the form of a chronic rather than an acute type of disease. The animal appears very dull and listless, and often there is discolouration of the skin around the base of the ears and on the ears, inside the forearms and thighs and along the belly. This discolouration is not in a confluent mass but takes the form of hæmorrhagic spots. There is frequently observed a cough, but this is not a very reliable symptom, as very many healthy pigs will cough and many that are suffering from swine fever do not cough. The bowels are generally constipated at first; later they are relaxed, due no doubt to the irritation of the mucous membrane. There is also a degree of lameness, or apparent weakness in the loins which causes a staggering gait, the animal walking comparatively firmly on the fore legs with the hind limbs reeling as if partially paralysed. I consider this one of the most diagnostic symptoms, and when observed with some of the other symptoms I have mentioned I have no hesitation in pronouncing the case one of swine fever.

The conclusive diagnosis must be made from a post-mortem examination, and the finding of either the so-called swine-fever ulcer or the diphtheritic inflammation of the alimentary canal. I propose here to consider those conditions that will lead us to diagnose the disease during life.

One naturally makes enquiries as to the history of the pigs, and when we find that they have been recently purchased or exposed for sale in an open market it makes us suspicious. But we may hear that they have been on the same premises for some months—from two to six months, but have been what are described as "bad doers." This is an almost constant accompaniment of swine fever. We may also find that all the pigs on the premises were bred there, but on further enquiry elicit the fact that some of them had visited other premises for stud purposes. I once had four cases like this in quick succession, and infection was traced to the boar.

There is no doubt that boars and sows will carry the disease for a considerable period without developing any apparent symptom but still be the means of infecting many other pigs.

As regards the discolouration of the skin, in swine fever this is of a scarlet crimson colour and found, as I have mentioned, about the ears and those parts where the skin is thin and comparatively free from hair. Though of a similar colour to that seen in cases of brine poisoning it should not be mistaken for that ailment, for in brine poisoning the discolouration is much more general and not confined to parts I have mentioned. Discolouration of the skin occurs also by the effects of the sun's rays, or "scorching." In these cases the discolouration is fairly general over the ears and back of the head, whereas in cases of swine fever it is deepest at the base of the ears and gradually spreads from there to the tips. The discolouration seen in cases of swine erysipelas or purples is, as the name implies, of a much darker and bluer tint, and is seen more evenly distributed over the surface of the body, though it may be patchy, but then the patches of discolouration appear to be somewhat raised above the level of the parts not so affected.

In styes where cases of swine fever exist there is generally to be observed a sour smell which is somewhat diagnostic. It is comparatively easy to recognise but difficult to describe. It resembles the odour of stale and very sour brewer's grains. It is my experience that swine fever seldom attacks with the same virulence all the pigs exposed to the same infection, for it is seldom one visits a herd of swine and finds many dead unless they are very young pigs. All *may* be suffering from the disease, but in the case of say 20, all apparently exposed to the same infection, there may be two or three dead and two or three more shewing symptoms of the disease, and if the remainder were put in quarantine for an indefinite period, as were the methods of the Board of Agriculture some two years ago, it was then found that many would linger on for many weeks while some would apparently not become affected at all.

In cases of swine fever the bowels invariably become relaxed, but in cases of swine erysipelas they are generally constipated.

In swine erysipelas and in porcine pneumonia the temperature runs to 107-108 (about), which is higher than in swine fever. Also in these diseases it falls as the cause is removed, whereas in swine fever as the disease becomes more intense or is further developed the temperature falls and very often is below normal long before death.

Recovery from swine fever I have had no experience of, and until I have seen cases in which the so-called cicatries of the swine fever ulcers are well marked I cannot believe that pigs do recover from the disease.

The staggering gait of pigs affected with swine fever can easily be distinguished from cases of traumatic injurices or from those suffering from rheumatoid arthritis, and needs no explanations from me, except it be to say that in the case of swine fever there is no apparent local lesion to account for the extreme lameness and loss of power, whereas in the other the evidence is generally apparent.

(To be continued.)

Alleged Cruelty by a V.S. at Chesterfield.

At Chesterfield, on Saturday, a charge of alleged cruelty to a horse was heard, in which Frank L. Somerset, veterinary surgeon, Holywell Street, was summoned by the Royal Society for the Prevention of Cruelty to Animals, for aiding and abetting, and counselling George Allen, farmer, Noth Wingfield, to cause a mare to be worked while in an unfit state. Mr. Ellison defended. The animal had fibroid tumours, and the prosecution alleged that pain must result from working it. Mr. Somerset denied this, and said all his tests proved that no pain was experienced.

The case occupied six hours and was adjourned.

On Monday, Nov. 23, a number of expert witnesses were called on the point as to whether or not pain was experienced by the mare from the fibroid tumours from which she was suffering, and the evidence was of a very conflicting character. It was alleged that Mr. Somerset had advised Allen that the mare could be worked, as although there might be blood no pain would result.

On behalf of Mr. Allen several witnesses were called, including men who had worked the mare, and the blacksmith who shod her, to prove that the defendant had worked her in the belief that there would be no pain.

The Chairman, at the close of the evidence, said the Bench had given the case very full consideration with the object of having everything made fair and plain. They had decided to convict Allen for the offence on the 15th October, and Wheatly, a servant who was a party to the proceedings, would only have to pay the costs, as

he had simply obeyed the orders of his master. Allen was fined £1 and costs, the Chairman stating that the penalty would have been heavier, but for the fact that he had called in professional advice after what had occurred on the 15th ult. The charge of causing the mare to be worked on the 25th, another date specified, would be dismissed.

With regard to Mr. Somerset, the matter was really one of professional opinion, and it was perhaps a pity that he and the Society's inspector did not meet and consult. In that way possibly these unpleasant proceedings might have been avoided. The Chairman added that he might be allowed to throw out a suggestion that a consultation should take place on any similar occasion.

Mr. Somerset: I should be delighted at the opportunity

Inspector Watkins: And so should I.

The Chairman: Well, we have had professional evidence on both sides, and shall dismiss the case against Mr. Somerset. We cannot help thinking it would be a monstrous thing, and not calculated to further the ends of justice, if a veterinary surgeon or a doctor could not prove his opinion, whether right or wrong, in a perfectly fearless manner and without a thought of a conviction. With regard to all the cases on both dates the Bench considered the Society was justified in bringing them before the Court.

Mr. Walker asked if it was to be understood that the mare could not be used for light work on the farm.

The Chairman: That matter is not before us. For my own part I do not mind telling you that I should not work the animal again.

THE MEMORIAL TO PROF. NOCARD.

Sir,—I have the satisfaction to announce that I have now received the sum of £105 towards the Nocard Memorial Fund, which is now closed, and I shall feel very much obliged if you will publish the enclosed further list of contributors.—Yours truly,

Dec. 3, 1903. ALEX. C. COPE.

The Right Hon. The Earl of Onslow, President Board of Agriculture and Fisheries.

Sir Thos. Henry Elliott, K.C.B., Secretary Board of Agriculture and Fisheries.

W. A. Byrne, Roscommon	Lt. W. A. Pallin, A.V.D.
Lieut. J. T. Coley, A.V.D.	G. Parr, Salisbury
F. P. Carter, Bradford	J. M. Parker, Birmingham
H. J. Dawes, W. Bromwich	R. C. Robinson, Carlisle
Maj. G. Elphick, Newcastle	W. W. Reekie, London
E. R. Edwards, Board of Agriculture	J. Storrar, Chester
E. Faulkner, Manchester	H. Sessions, Newport, Mon.
G. H. Harris, Sawbridgeworth	C. Stephenson, Newcastle
H. Jarvis, St. Leonards	R. C. Trigger, Newcastle-under-Lyme
D. Pugh, Sevenoaks	J. B. Wolstenholme, Manchester
	F. W. Wragg, London

ERRATUM.—For A. Goodall (last week) read J. B. Goodall.

OBITUARY.

JOHN THOMAS KING, M.R.C.V.S., Bournemouth, Hants. Graduated, Lond: Dec. 1873.

We regret to record the somewhat sudden death of Mr. J. T. King, which occurred at his residence, 14 Southcote Road, on Monday, Nov. 16. Mr. King, who was 62 years of age, never thoroughly recovered from an attack of influenza from which he suffered some ten years ago, an affection of the heart being left. To the ordinary observer, however, he continued in sound health, and as active as ever in a very industrious and useful career. Within the last three weeks he showed symptoms of rapidly failing health, and for a fortnight kept his bed, suffering from an acute form of muscular rheumatism. Though his condition was serious, there was nothing to occasion alarm until early on Monday morning, when it was found that the patient had taken a turn for the worse. He sank rapidly and died about a quarter past nine o'clock.

Mr. King had held the post of Veterinary Surgeon to the governing body of Bournemouth from 1874, previous to which date he was for some years the Consulting Veterinary Surgeon to the town and to the old Board of Commissioners. He was the local Veterinary inspector to the L. & S.W.R. Co., and also for many years Veterinary Surgeon to the Royal Society for the Prevention of Cruelty to Animals. During the late South African War he undertook the transport of a number of remounts. On his return he related his experiences to a public meeting in Bournemouth, and was one of those who championed the action of the War Office. Mr. King was of a kindly but quiet disposition, and was liked by all who knew him. He was peculiarly adapted to his profession, the duties of which he discharged with much ability, and the claims upon his services, though always numerous, were ever responded to with cheerfulness owing to a true love for the dumb creation and an earnest desire to relieve suffering. By his death the town has lost a worthy burgess, and the services of a very able practitioner. He leaves a widow but no family.

The funeral was attended by the Mayor and Corporation and Borough Officials, Messrs. T. B. Goodall, (Christchurch), E. Whitley Baker (Wimbourne), and Pack (Lymington) (representatives of the Southern Counties Veterinary Medical Association), Mr. T. Aubrey, and a number of others.—*The Bournemouth Directory.*

ARMY VETERINARY DEPARTMENT.

Extract from *London Gazette*,

WAR OFFICE, PALL MALL, Nov. 27.

The undermentioned Lieutenants to be Captains:—E. Brown, R. F. St. C. Houston.

Dec. 1.

The undermentioned Captains to be Majors:—Thomas Emmanuel Watkins Lewis, Eugene William Larnder.

CORRESPONDENCE.

MILK FEVER.

Sir,

After perusing "Lincs." dolorous account of his experience of milk fever, and calling to memory the words of my esteemed teacher, Prof. Mettam, "that some animals will recover in spite of care," I marvelled at his high percentage of fatalities. The auspices of the Gods have apparently been against "Lincs." even thongh he did wrap his patients in sheep's clothing and tried a host of remedies. One naturally comes to Mr. Mayall's conclusion, that although unsuccessful with Schmidt's treatment, "Lincs." has failed to substitute anything better.

My experience of the disease has been a little more pleasant. During the past season Mr. Olver and I have treated about 40 cases with only four deaths, three of these were tympanitic and too far gone before medical aid was called in, while the fourth dropped down and suddenly expired after recovering from a second attack. The Chinosol treatment is the one adopted, 50 to 60 grs. are dissolved in about a quart of lukewarm water and a fourth part injected into each quarter of the udder along with air, the udder having been previously milked perfectly dry. I find it advantageous to use a good quantity of water and then to distend the udder well with air, massaging gently afterwards. A second injection is sometimes necessary in very bad and lingering cases, followed up with Chloral

hydrate given in ʒss. doses in draughts every two hours. I may say that I have found Epsom salts detrimental to a rapid recovery, the four fatal cases mentioned above all received large doses of salts.

With regard to Mr. Hudson's stimlant treatment, I think it is contra-indicated in the early stages of the disease, and in my opinion ought not to be given until the animal has well recovered consciousness, as I have sometimes brought on a second attack by giving stimulants too soon. The smothering system I also take objection to, as many of the deaths after lung affections arise from too frequent drenching and the exclusion of fresh air, Nature's Balm of Gilead. I have used the above treatment on all sorts of breeds, from the biggest Shorthorn to the smallest Jersey. I have used it near John-o'-Groats and used it near Land's End, the results being pretty much the same, only I prefer Chinosol to Pot. iodide, as the former being a better antiseptic lessens the danger of mammitis from bacterial contamination.

I do not contend, nor even hope to be as successful every season with Schmidt's treatment, as a great deal depends on your early presence on the scene of operation and other unforseen circumstances, but it will take more than a sheep skin or rug smothering theory to cause me to give it up under our present knowledge of the disease.

JAMES FORBES, M.R.C.V.S.

Truro.

ACCIDENT INSURANCE COMPANIES AND VETERINARY SURGEONS.

Sir,

Having lately been the subject of what I consider grossly unfair treatment by an Accident Insurance Company, I think it is only fair to warn veterinary surgeons generally what to expect under similar circumstances.

I have been insured with a company for upwards of twenty years under the usual conditions of £1000 at death, £6 per week during total disablement from work, and 30s. per week for partial. Veterinary surgeons pay on the higher scale, my premium being £5 10s. per annum.

On the 19th June last, I accidentally ruptured some fibres of the psoas muscle, I was in bed for two weeks unable to move, at the end of which time my doctor consented to my being carried up and down stairs, and with the help of crutches was able to get about a few yards, and sit or lie down, this continued for the next two or three weeks. I then gladly changed the crutches for two stout sticks, and later to one stick. All this time it would have been quite impossible for me to get into my trap for business or pleasure, quite apart from any question of suffering.

I claimed seven weeks and two days "total" and four weeks "partial" disablement. The Company having considered my claim most generously awarded me *two* weeks "total" and considered that from that time I was able to partially attend to my professional duties. My doctor explained how necessary "agility" was in a V.S., the serious risks I should have run in doing as they suggested, had it been possible, but with no further success than an offer to halve the amount in dispute, about £18, (thus allowing me equal to another one and a half weeks' "total" rate of payment) or submit the whole matter to arbitration, a somewhat risky and costly proceeding.

Of course the whole question turns on *when* a veterinary surgeon (whose duties are mostly with more or less unruly patients) is to be *fairly considered* as "totally incapacitated" from pursuing his usual professional duties.

I have no desire for anything but what is fair and right, but I was under the impression I was insured properly, for my then condition, which I submit was *most certainly* one of total incapacity *for a veterinary surgeon*, but have found to my cost such was not the case.

To an active man who loves his profession I know of no greater trial than to be unable to get about as usual and see clients etc., but if one employs a qualified *locum tenens* you must leave him to do the work his way, so long as you can trust him and know he is giving satisfaction, otherwise you worry yourself needlessly and retard your progress, besides being a nuisance to your *locum*.

If I could have got about, as the Insurance Company seem to imagine, I should have done my work myself, and certainly not have gone to the expense of a *locum* to fill my place for so long a period as I was obliged to do.—Yours faithfully,

F. MORTON WALLIS.

Halstead, Essex.

EXEMPTION FROM JURY SERVICE.

Sir,

A short time ago, when the Jury lists for this city were being revised, I instructed my solicitor to claim exemption for me, with the result, I am pleased to say, that it was allowed in the case of assizes and sessions. I am informed all persons are liable to serve on a coroner's jury, however, out of courtesy, my name was also removed from that list. I trust universal exemption for members of our profession will be established in the near future, as I know jury service has inconvenienced many of our members, to say nothing of the elevation in the social scale of our profession, which will be evident to the general public. While agreeing with views expressed by yourself on previous occasions "that it is desirable a supply of educated, intelligent men should be available for jury service," I fail to see why a small, hard-working profession like ours should have to bear the burden of State service, while others, no more useful and deserving of consideration, are exempted.

JOHN BLAKEWAY.

Birmingham, 30th Nov., 1903.

DISEASES OF POULTRY.

Sir,

In reply to your correspondent Galen's inquiry about a book on diseases of poultry, perhaps the following may be of some use to him.

1. Traité de l'Elèvage et des Maladies des Animaux et Oiseaux de basse-cour, by M. ,Benion, published by Asselin & Houzeau, place de l'Ecole de Médecine, Paris. 7 francs.

2. Les Oiseaux de Basse Cour, by Remy Saint Loup. 368 pages, 105 illustrations. 4 francs.

3. Canards, Oies et Cygnes, by A. Blanchon. 348 pages, 73 illustrations. Price 4 francs.

The last two are published by J. B. Baillière et fils, 19 Rue Hautefeuille, Paris.—Yours,

JOSHUA A. NUNN, F.R.C.V.S.

A CORRECTION.—DISLOCATION OF THE FETLOCK.

We regret that an error occurred in the name of the author of this note (p. 306).

It should read J. FENN ADDISON, and not as printed.

COMMUNICATIONS, BOOKS AND PAPERS RECEIVED.—Messrs. G. M. Pomery, Gerald Leighton, M.D., J. Forbes, R. Jones, "Galen." *Parliamentary Debates (N.Z.)* *No. 24.* Nov. 28.

Messrs. W. Pauer, George Upton, R. E. Montgomery, F. M. Wallis, John Blakeway, A. C. Cope. Col. J. A. Nunn. G. M. Dec. 5.